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Network structure of depression symptomology in participants with and without depressive disorder: the population-based Health 2000–2011 study

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Abstract

Purpose Putative causal relations among depressive symptoms in forms of network structures have been of recent interest, with prior studies suggesting that high connectivity of the symptom network may drive the disease process. We examined in detail the network structure of depressive symptoms among participants with and without depressive disorders (DD; consisting of major depressive disorder (MDD) and dysthymia) at two time points.

Methods Participants were from the nationally representative Health 2000 and Health 2011 surveys. In 2000 and 2011, there were 5998 healthy participants (DD-) and 595 participants with DD diagnosis (DD+). Depressive symptoms were measured using the 13-item version of the Beck Depression Inventory (BDI). Fused Graphical Lasso was used to estimate network structures, and mixed graphical models were used to assess network connectivity and symptom centrality. Network community structure was examined using the walktrap-algorithm and minimum spanning trees (MST). Symptom centrality was evaluated with expected influence and participation coefficients.

Results Overall connectivity did not differ between networks from participants with and without DD, but more simple community structure was observed among those with DD compared to those without DD. Exploratory analyses revealed small differences between the samples in the order of one centrality estimate participation coefficient.

Conclusions Community structure, but not overall connectivity of the symptom network, may be different for people with DD compared to people without DD. This difference may be of importance when estimating the overall connectivity differences between groups with and without mental disorders.

Keywords Network · Connectivity · Depression · Symptoms

Introduction

Depressive disorders (DD), including major depressive disorder (MDD) and dysthymia, are highly prevalent mental disorders with high comorbidity with other mental disorders. Although they have been under systematic investigation for decades, depressive disorders remain poorly understood, and treatment efficacy has been modest [1]. It has been traditionally assumed that depressive symptoms arise from common pathogenic pathways. Recently, this common cause-approach has been challenged [2] by research showing that different depressive symptoms are associated with different risk factors [3], different patterns of comorbidity [6], and are associated with different levels of impairment [3]. Consistent with the above evidence of differential relations between symptoms and varying outcomes, depression

